AMENDMENTS TO THE CLAIMS

1. (Currently amended) A method of modulating endothelial cell activity, said method comprising modulating the functional activity of protein kinase Cζ, wherein up-regulating protein kinase Cζ activity to a functionally effective level up-regulates said cellular activity and down-regulating protein kinase Cζ activity to a functionally ineffective level down-regulates said cellular activity.

2. (Previously presented) The method according to claim 1, wherein said endothelial cell is a vascular endothelial cell or a lymphatic endothelial cell.

3. (Previously presented) The method according to claim 1, wherein said cellular activity is endothelial cell permeability.

4. (Previously presented) The method according to claim 3, wherein said endothelial cell permeability is intercellular or intracellular.

5. (Previously presented) The method according to claim 4, wherein said permeability is thrombin-induced vascular endothelial cell permeability.

6. (Previously presented) The method according to claim 1, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by introducing into said endothelial cell a nucleic acid molecule encoding protein kinase Cζ or functional equivalent, derivative or homologue thereof or the protein kinase Cζ expression product or functional derivative, homologue, analogue, equivalent or mimetic thereof.

7. (Currently amended) The method according to claim 1, wherein said modulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which modulates transcriptional and/or translational regulation of the protein kinase Cζ gene.

8. (Currently amended) The method according to claim 1, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which functions as an agonist of the protein kinase Cζ expression product.

9. (Currently amended) The method according to claim 1, wherein said modulation is down-regulation of protein kinase Cζ activity and said down-regulation is achieved by

contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~modulation agent, which functions as an antagonist to the protein kinase Cζ expression product.

10. (Currently amended) The method according to claim 9, wherein said ~~molecule~~modulation agent is angiopoietin-1 or functional derivative, homologue, analogue, equivalent or mimetic thereof.

11. (Currently amended) The method according to claim 9, wherein said ~~molecule~~modulation agent is chelerythrine chloride or bisindoylmaleimide I or functional derivative, homologue, analogue, equivalent or mimetic thereof.

12. (Currently amended) The method according to claim 9, wherein said ~~molecule~~modulation agent is a mutant protein kinase Cζ, which mutant is characterised by substitution of the threonine residue at position 410 of the activation loop to alanine.

13. (Previously presented) The method according to claim 1, wherein said endothelial cell activity is modulated *in vivo*.

14. (Previously presented) The method according to claim 13, wherein said endothelial cell activity is modulated *in vitro*.

15. (Original) A method of regulating endothelial cell activity in a mammal, said method comprising modulating the functional activity of protein kinase Cζ in said mammal wherein up-regulating protein kinase Cζ activity to a functionally effective level up-regulates said endothelial cell activity and down-regulating protein kinase Cζ activity to a functionally ineffective level down-regulates said endothelial cell activity.

16. (Previously presented) The method according to claim 15, wherein said endothelial cell is a vascular endothelial cell or a lymphatic endothelial cell.

17. (Previously presented) The method according to claim 15, wherein said cellular activity is endothelial cell permeability.

18. (Previously presented) The method according to claim 17, wherein said endothelial cell permeability is intercellular or intracellular.

19. (Previously presented) The method according to claim 18, wherein said permeability is thrombin-induced vascular endothelial cell permeability.

20. (Previously presented) The method according to claim 15, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by

introducing into said endothelial cell a nucleic acid molecule encoding protein kinase Cζ or functional equivalent, derivative or homologue thereof or the protein kinase Cζ expression product or functional derivative, homologue, analogue, equivalent or mimetic thereof.

21. (Currently amended) The method according to claim 15, wherein said modulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which modulates transcriptional and/or translational regulation of the protein kinase Cζ gene.

22. (Currently amended) The method according to claim 15, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which functions as an agonist of the protein kinase Cζ expression product.

23. (Currently amended) The method according to claim 15, wherein said modulation is down-regulation of protein kinase Cζ activity and said down-regulation is achieved by contacting said endothelial cell with a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which functions as an antagonist to the protein kinase Cζ expression product.

24. (Currently amended) The method according to claim 23, wherein said ~~molecule~~modulation agent is angiopoietin-1 or functional derivative, homologue, analogue, equivalent or mimetic thereof.

25. (Currently amended) The method according to claim 23, wherein said ~~molecule~~modulation agent is chelerythrine chloride or bisindoylmaleimide I or functional derivative, homologue, analogue, equivalent or mimetic thereof.

26. (Currently amended) The method according to claim 23, wherein said ~~molecule~~modulation agent is a mutant protein kinase Cζ which mutant is characterised by substitution of the threonine residue at position 410 of the activation loop to alanine.

27. (Original) A method for the treatment and/or prophylaxis of a condition characterised by aberrant, unwanted or otherwise inappropriate endothelial cell activity in a mammal, said method comprising modulating the functional activity of protein kinase Cζ wherein up-regulating protein kinase Cζ activity to a functionally effective level up-regulates said endothelial cell activity and down-regulating protein kinase Cζ activity to a functional ineffective level down-regulates said endothelial cell activity.

28. (Previously presented) The method according to claim 25, wherein said endothelial cell is a vascular endothelial cell or lymphatic endothelial cell.

29. (Previously presented) The method according to claim 25, wherein said cellular activity is endothelial cell permeability.

30. (Previously presented) The method according to claim 29, wherein said endothelial cell permeability is intercellular or intracellular.

31. (Previously presented) The method according to claim 30, wherein said permeability is thrombin-induced vascular endothelial cell permeability.

32. (Previously presented) The method according to claim 27, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by introducing to said mammal a nucleic acid molecule encoding protein kinase Cζ or functional equivalent, derivative or homologue thereof or the protein kinase Cζ expression product or functional derivative, homologue, analogue, equivalent or mimetic thereof.

33. (Currently amended) The method according to claim 27, wherein said modulation is achieved by introducing to said mammal a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which modulates transcriptional and/or translational regulation of the protein kinase Cζ gene.

34. (Currently amended) The method according to claim 27, wherein said modulation is up-regulation of protein kinase Cζ activity and said up-regulation is achieved by introducing to said mammal a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which functions as an agonist of the protein kinase Cζ expression product.

35. (Currently amended) The method according to claim 27, wherein said modulation is down-regulation of protein kinase Cζ activity and said down-regulation is achieved by introducing to said mammal a proteinaceous or non-proteinaceous ~~molecule~~ modulation agent which functions as an antagonist to the protein kinase Cζ expression product.

36. (Currently amended) The method according to claim 35, wherein said ~~molecule~~modulation agent is angiopoietin-1 or functional derivative, homologue, analogue, equivalent or mimetic thereof.

37. (Currently amended) The method according to claim 35, wherein said ~~molecule~~modulation agent is chelerythrine chloride or bisindoylmaleimide I or functional derivative, homologue, analogue, equivalent or mimetic thereof.

38. (Currently amended) The method according to claim 35, wherein said ~~molecule~~modulation agent is a mutant protein kinase Cζ, which mutant is characterised by substitution of the threonine residue at position 410 of the activation loop to alanine.

39. (Previously presented) The method according to claim 29, wherein said condition is an inflammatory response.

40. (Previously presented) The method according to claim 29, wherein said condition is unwanted angiogenesis.

41. (Previously presented) The method according to claim 40, wherein said condition is solid tumors, blood born tumors, tumor metastasis, benign tumors, rheumatoid arthritis, Crohn's disease, atherosclerosis, obesity, endometriosis, ocular angiogenic diseases, psoriasis, facial and truncal telangiectasias, or Osler-Webber Rendau syndrome.

42-48. (Canceled).

49. (Currently amended) The method of claim 9, wherein said endothelial cell activity is endothelial cell intercellular permeability, and wherein said endothelial cell is contacted with a small molecule ~~inhibitor~~ antagonist of the PKCζ expression product.

50. (New) The method of Claim 9, wherein said modulation agent is selected from the group consisting of a small molecule, an antibody, or an analogue of the PKCζ expression product.

51. (New) The method of Claim 7, wherein said modulation agent is selected from the group consisting of antibodies, antigens, RNA, ribosomes, DNAzymes, RNA aptamers, and antisense nucleic acids.